Successful Closure of a Giant True Saphenous Vein Graft Aneurysm Using the Amplatzer Vascular Plug

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An 85-year-old man was found to have a 9 cm diameter true aneurysm of an obtuse marginal saphenous vein graft. The graft was fed by a relatively narrow neck from the proximal remnant of the obtuse marginal graft 10 years after prior coronary artery bypass graft surgery. An Amplatzer vascular plug was used successfully to occlude the neck of the aneurysm. Follow-up CT scan 1 month afterward demonstrated retained contrast in the aneurysm, demonstrating complete occlusion between the aorta and aneurysm sac. Pressure recordings from within the aneurysm sac suggest bidirectional flow in the neck as the mechanism for the maintained patency of the aneurysm. The use of a vascular plug is an effective method for aneurysm occlusion when the anatomy is suitable.

INTRODUCTION

Giant true aneurysms of coronary saphenous venous grafts (SVGs) are a rare complication of coronary artery bypass surgery, first reported by Riahi et al. [1] in 1975. In contrast, mild dilatation of saphenous vein grafts is not uncommon and has been estimated to occur in roughly 14% of cases after 5–7 years after coronary artery bypass graft surgery [2]. Significant dilatation associated with true aneurysms, classified as greater than 3 cm in diameter [3], has been reported in fewer than 20 cases [4,5]. One review by Memon et al. [2] cited 50 cases of true aneurysms of any diameter, including cases less than 3 cm. Potential complications may include myocardial infarction, embolization, angina, heart failure, arrhythmias, compression, and rupture leading to life-threatening hemorrhage. Most reports of SVG aneurysms have advocated surgical removal, while a few authors have used coil embolization [6,7] or exclusion with vein-covered stents [8]. We present a case of an SVG giant true aneurysm with successful occlusion using an AGA Medical Amplatzer vascular plug.

CASE REPORT

An 85-year-old male was referred for evaluation of atrial fibrillation and possible radiofrequency catheter ablation. Over the previous 2 months, the patient had been hospitalized for intermittent atrial fibrillation with rapid ventricular response refractory to medical treatment, in addition to symptoms of fatigue and malaise. His past medical history was significant for myocardial infarction 30 years ago, multiple near syncopal episodes requiring recent dual-chamber pacemaker placement for sick sinus syndrome, and coronary artery bypass graft surgery (LIMA to LAD, SVGs to RCA and OM) and St. Jude prosthetic mitral valve replacement in 1994.

A chest radiograph demonstrated a mediastinal mass located on the left heart border (Fig. 1). Transesophageal echocardiography showed an abnormality thought initially to be a ventricular pseudoaneurysm (Fig. 2). A CT without contrast showed a large heterogeneous mass measuring $8.7 \times 9.2 \text{ cm}$ located at the level of the mitral valve between the left atrium and left ventricle along the left cardiac border (Fig. 3). The patient underwent coronary angiography, which demonstrated a patent LIMA to LAD, patent SVG to RCA, totally occluded SVG to OM at its origin, and a second SVG to OM feeding a large aneurysmal sac (Fig. 4). Additional findings included elevated right-sided heart pressure (RV 46/7/9 mm Hg, PA 42/14/28 mm Hg). The pulmonary artery...
saturation was 57%, which confirmed that there was no apparent shunt between the SVG aneurysm and the pulmonary circulation.

A decision to occlude flow to the aneurysmal sac was made 1 week after the initial catheterization demonstrated the SVG aneurysm. The patients’ warfarin therapy was interrupted. The OM SVG was engaged using a 7 Fr AL1 guide and a RADI pressure wire was used to measure pressures in the aneurysm sac (Fig. 5). Rest pressures within the aneurysm sac were 125/87 mm Hg with a mean pressure of 95, while those at the aortic orifice were 140/69 mm Hg with a mean pressure of 94. Anticoagulation with heparin was given with an ACT of 245 sec. A single 8 mm AGA Medical vascular plug (AGA Medical, Golden Valley, MN) was placed in the neck segment of the SVG (Fig. 6). Angiography demonstrated complete cessation of flow into the aneurysm (Fig. 7). The patient was discharged the next day without any complications. A follow-up CT performed approximately 4 weeks later to evaluate a complaint of chest pain without ECG or enzyme abnormalities demonstrated retained contrast within the SVG aneurysm suggesting complete occlusion between the aorta and aneurysm sac (Fig. 8). It was speculated that the chest pain may have been related to thrombosis of the aneurysm.

DISCUSSION

Large saphenous vein graft aneurysms are a rare complication of coronary artery bypass surgery usually presenting several years after the surgery [1–3,6,8–16]. Interestingly, there have only been two documented cases of SVG aneurysms occurring within 6 months after CABG [1,9]. Different mechanisms for the development of SVG aneurysms have been proposed. Generally, aneurysms of SVG appearing several years after the surgery are attributed to hyperlipidemia, atherosclerosis, hypertension [2,6,13–15,17,18], and undetected preexisting venous varicosities of the grafted vessel [19,20]. It has been suggested that the site of venous valves in the graft, where the transition between circular and longitudinal muscular layers in the vessel wall occurs, could be responsible for early SVG aneurysm formation [14,21].

These mechanisms offer no explanation for the hemodynamic and anticoagulant circumstances that must be
present for an aneurysm to develop and not spontaneously thrombose. In the case we present, the patient had been on chronic warfarin therapy for atrial fibrillation. It is also clear that there was bidirectional flow in the aneurysm neck. This is apparent since contrast cleared from the aneurysm sac after the diagnostic catheterization, and because spontaneous thrombosis did not occur.

Pressure recorded from within the sac may elucidate the mechanism for bidirectional flow in the neck. The pressure recording (Fig. 5) appears to have excluded transmitted pulsations from the lungs, since there are no respirophasic gradients between the central aortic and sac pressures. Streaming is noted in some of the angiographic images of the aneurysm neck (Fig. 9). This

Fig. 3. Computed tomogram of the chest without contrast obtained shortly after the chest X-ray diagnosis suggested a mass. A–D show the extracardiac mass denoted by an asterisk on successive tomographic cuts. There is a layer of thrombus within the sac.

Fig. 4. A: Right anterior oblique views from coronary angiograms of the saphenous vein graft feeding the aneurysm sac. The neck of the graft near its origin is moderately narrowed and the graft becomes ectatic further along its course. The large aneurysm sac can be seen to fill slowly on successive angiographic frames. B: Left anterior oblique angiographic images show the length of the saphenous vein graft with moderate diffuse disease and contrast filling the irregularly shaped large mass in the left thorax. This image is a composite of two angiographic frames.
prominent streaming suggests that there may be currents or areas of bidirectional flow within the neck of the aneurysm sac. Figure 5 shows that the aortic systolic pressure is higher than the aneurysm systolic pressure, and the aortic diastolic pressure is lower than the aneurysm diastolic pressure. Thus, there are gradients in either direction at some point during the cardiac cycle. This would appear to favor flow into the aneurysm during systole and flow back into the aorta during diastole. AO, aortic pressure; SVG, aneurysm sac pressure recorded from the pressure wire.

SVG aneurysms represent a clinical and diagnostic challenge. Based on one author’s recent review of the literature, roughly 50% of patients are asymptomatic at presentation with a mass detected on routine chest radiography; the rest of patients present with myocardial infarction, unstable angina, or heart failure in descending order of incidence [2]. It is crucial to detect these rare

Fig. 5. Simultaneous pressure records from the guide catheter in the aortic root and a pressure wire placed deep in the aneurysm sac. The aortic systolic pressure is higher than the aneurysm systolic pressure, and the aortic diastolic pressure is lower than the aneurysm diastolic pressure. Thus, there are gradients in either direction at some point during the cardiac cycle. This would appear to favor flow into the aneurysm during systole and flow back into the aorta during diastole. AO, aortic pressure; SVG, aneurysm sac pressure recorded from the pressure wire.

Fig. 6. This schematic shows an Amplatzer plug device delivered into an artery via a guide catheter. Between the end of the guide catheter and the base of the plug is the delivery cable, which attaches to the plug by a screw mechanism similar to the AGA Medical shunt closure devices. Image used with permission from AGA Medical Corporation.

Fig. 7. The top panel shows a guide catheter engaged in the graft origin. The white arrow points at the marker tip of the vascular plug. The guide catheter has been deeply seated in the saphenous vein graft. In the bottom panel, contrast no longer passes beyond the vascular plug. The plug has been detached and the proximal and distal markers are seen in the mid graft.
phenomena early in their course as some potential complications that may occur include embolization [20], myocardial infarction, compression or fistulization with other vascular structures [1,6,12,14,22], and rupture with hemothorax or hemopericardium leading to sudden death [2,5,23].

Traditionally, the majority of reports have advocated that true SVG aneurysm be treated with surgical excision or resection. A disadvantage of surgery is that it is at least a second sternotomy, with increased morbidity and mortality risk [22,24]. Coil embolization has been used as an interventional alternative in two cases [6,7]. Multiple coils must be used to occlude the vessel inevitably prolonging fluoroscopic procedure times. This approach also carries the risk of misplacement, dislodgement, or embolization of the coils [20,22]. One report described the use of a vein-covered stent to treat a small or moderate-sized aneurysm [8].

The Amplatzer vascular plug used successfully to occlude the SVG aneurysm in our case is a self-expanding cylindrical device made from a nitinol wire mesh and secured on both ends with platinum/iridium marker bands (Fig. 6). A stainless steel microscrew is attached to one of the platinum markers, allowing a delivery cable to be connected to the device. Sizes range from 4 to 16 mm in 2 mm increments. The thickness of mesh wire is 0.0015–0.003". The plug measures 7 mm in length for diameter sizes 4–10 mm and 8 mm for sizes 12–16 mm. The delivery cable is 0.032” in diameter, in contrast to the ASD occluder which has a 0.075” diameter delivery cable and the PDA device with a 0.044” cable. After the Amplatz vascular plug has been preloaded in a loader, it can be delivered using either a 4 or 5 Fr Mullins-type sheath or through 5–8 Fr guide catheters, depending on the size of the vascular plug used. Unlike the PDA and ASD devices, the vascular plug does not have an internal polyester fabric component. The vascular plug chosen should be roughly 20–30% larger than the vessel to be occluded. Currently, the Amplatz vascular plug is indicated for arterial and venous embolizations in the peripheral vasculature. This single device can be placed within a target vessel, repositioned if necessary, and finally deployed in a precise and controlled manner. In addition, the nitinol wire frame provides favorable radial traction that secures it within the vessel wall, eventually becoming endothelialized and thrombosed, providing complete occlusion of the target vessel.

Recently, a case report has documented the successful closure of aortopulmonary collaterals arising from the descending thoracic aorta in a 4.5-month-old infant using the Amplatz plug [25]. In our case, the SVG
aneurysm’s dimensions had favorable size and morphology for this device. Nevertheless, the safety and effectiveness of this device for cardiac and neurological uses has not been established. Potential complications noted by the manufacturer included site of entry hematoma, vessel perforation, and embolization of the device.

We present a method for successfully occluding a giant true SVG aneurysm using the Amplatzer vascular plug. Given the wide selection of sizes available, the geometry, the ability to reconstrain and reposition the device, and the maneuverability allowing precise placement, we believe this device is well suited for treating this condition. This represents a viable alternative to surgery or coil embolization. The long-term durability of this approach is yet to be assessed.

REFERENCES